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Considerations in Assessmemt and Care of the High Risk Neonatal Foal

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It is absolutely critical to recognize and assess a sick foal early in the course of disease. There may be several predisposing conditions, before and after birth, that put a foal into the high risk category. Conditions such as purulent vaginal discharge, colic surgery, general anesthesia, endotoxemia, poor nutritional status and excessive colostral leakage prior to parturition are a few maternal conditions that may adversely affect an unborn foal. Adverse conditions of parturition and specific neonatal complications may also put a foal at high risk, including premature parturition, prolonged labor or induction of labor, dystocia, placental abnormalities, twinning, orphan, delay or lack of intake of sufficient quantities of colostrum, immaturity or prematurity and exposure to infectious diseases.[1]

A thorough initial work-up of the neonatal foal is essential. Many foals present with vague clinical signs such as weakness and decreased appetite while others may be comatose and near death. Any delay in initiating therapy may make the difference between a live or dead foal. Most abnormal neonatal foals rarely have one problem. The sicker they are, the more likely they are to develop a series of problems.[1]

Regardless of the inciting cause, a critically ill neonatal foal's most common problem is infection. One of the most common causes of infection is the result of failure of the foal to acquire adequate colostral immunity.[3]

The mammary gland of the mare concentrates immunoglobulin from the blood, primarily IgG with smaller amounts of IgA and IgM. The secretion of colostrum lasts for a short period of time and is replaced by milk which has much lower concentrations of immunoglobulin.[3] Colostrum also provides lymphocytes and factors which exert local protective effects on the gastrointestinal tract.

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The absorption of colostral immunoglobulin is due to the specialized epithelial cells in the small intestine that non-selectively take-up colostral immunoglobulin by pinocytosis.[1] The uptake of immunogloblins decreases with time with maximum absorption within 8 hours of parturition.[3] Because the specialized epithelial cells are replaced within 24-36 hours by more mature cells that are unable to take up colostral immunoglobulin (gut closure), it is very important the foal receive colostrum within 12 to 18 hours of birth and optimally within 6 hours for best absorption.[3] If a foal nurses and receives adequate colostrum within 2 hours of birth, serum IgG levels become detectable at about 6 hours of age and peak at about 18 hours of age.[1]

In addition, because the placenta of the mare is epitheliochorial, the transfer of maternal immunoglobulin in-utero to the fetus does not occur. As a result, the presuckle foal does not have protective immunoglobulin levels of its own against environmental pathogens to which it is exposed. The newborn foal is protected from disease for the first 4-8 weeks of life by passively transferred colostral immunoblobulins and by non-specific host defense mechanisms.[1]

Even though the foal is immunologically competent at birth, the presuckle foal is agammaglobulinemic.[1] T-lymphocytes are present by 100 days' gestation. B-lymphocytes produce immunoglobin by 180 days' gestation, but the number of circulating B-cells is about one third of those in the adult horse until the foal is about 3 weeks of age.[1] Because the immune system of the neonatal foal is naive, the primary immune response is not detectable as serum IgG until the foal is 2 weeks old.[1]

Immunoglobulins passively transferred are catabolized very quickly in the first 4 weeks of life and are diluted by an increasing plasma volume. Autogenous IgG can be detected by 2 weeks of age, but adult levels are not reached until about 4 months of age.[1] Also, foals that are deprived of colostrum will produce their own immunoglobulin much earlier than a foal fed adequate amounts of colostrum.

Failure of passive transfer (FPT) of immunoglobulin is thought to be the most common predisposing cause of infection in foals younger than 2 weeks of age.[1] The IgG level of a normal foal should be at least >800 mg/dl. Partial failure of passive transfer is considered at serum immunoglobulin levels of 200-400 mg/dl and total FPT at <200 mg/dl.

The causes of FPT are numerous, and include loss of colostrum before parturition (premature lactation), failure of the newborn foal to ingest an adequate amount of colostrum within the first 12 hours of life, inadequate IgG concentration in colostrum and insufficient IgG absorption by the intestinal epithelium.[3]

Depending on the types of bacteria present, management, stress factors, and colostral antibody titer against specific bacteria in the environment, the minimum level of IgG needed to protect the foal from infection may vary considerably. Most of the data collected has confirmed the high-risk foal should have an IgG concentration of greater than 800mg/dl to be protected.[1]

When assessing a foal for FPT, serum IgG levels should be re-evaluated between 6 to 12 hours of age, so that additional colostrum can be supplemented orally if IgG levels are less than 400 mg/dl. There are several tests available to rapidly assess IgG such as zinc sulfate turbidity test, the latex agglutination test and the CITE test. Although signs of septicemia secondary to FPT usually occur on the third to fourth day of life, a bacteremia may already be present at 24 hours of age or earlier.

When presented with a neonatal foal that is sick and depressed, there are several differentials that should be considered such as neonatal maladjustment syndrome, prematurity and immaturity, meconium retention, neonatal isoerythrocytolysis, and ruptured bladder (uroperitoneum).[5]

Neonatal maladjustment syndrome (NMS) is a condition of newborn foals (non-infectious) characterized by loss of suck reflex and bonding with mare, gross behavioral changes such as wandering, blindness, grinding teeth, extensor rigidity, opisthotonos, convulsions and sometimes coma.[5] The condition sometimes is accompanied by multiple organ system failure. The cause of NMS is unknown,but hypothesized to be secondary to a pre-parturient hypoxemia.[6] Foals with NMS usually remain strong and are not febrile, unless they become septic.[2]

Prematurity is defined as a foal born at a gestational age of younger than 320 days and shows immature physical characteristics such as generalized weakness and reduced suck reflex. These foals tend to have low birth weights, take a longer time to stand after birth, and develop decubital ulcers because of their thin "silky" skin.[5] Premature foals may become hypothermic and also develop atelectasis if they can notfully inflate their lungs. An immature/dysmature foal is a term newborn foal that shows signs of prematurity.[2]

Foals with retained or impacted meconium may show signs of straining or colic within a few hours of birth. The body temperature and cardiovascular status of these foals are normal.[2]

Neonatal isoerythrolysis is an isoimmune condition of foals caused by intravascular or extravascular hemolysis. These foals usually appear normal at birth and the first few hours of life. Clinical signs such as weakness, lethargy, icterus, pale mucus membranes, and hemoglobinuria then develop. The PCV and RBC counts of these foals are markedly decreased. Additionally, increased heart rate and respiratory rate, seizures, and absence of fever are seen. These signs usually appear at about 8 to 96 hours after birth.[1]

Male foals present with ruptured bladder and uroperitoneum more frequently than female foals. These foals are normal at birth, then start to dribble urine, strain to urinate, and develop abdominal distension. Blood chemistry profile reveals hyponatremia, hypochloremia, hyperkalemia, and a high ratio of peritoneal fluid creatine to serum creatinine.[5] These foals may have a severely decreased circulating blood volume due to the accumulation of fluid in the peritoneal cavity. Signs such as poor pulse quality, decreased peripheral perfusion, respiratory distress, and severe depression all may be noted depending on how advanced the condition is on presentation.

Early in clinical disease, septicemia can be difficult to diagnose because of the non-specific signs. Later, septicemia often presents with severe pneumonia, polyarthritis, osteomyelitis, diarrhea, or omphalitis. The presence of fever, septic shock, or lesions caused by blood-borne bacteria (meningitis, polyarthritis, uveitis) are good evidence for the presence of septicemia. The bacteria can easily spread from the lung, gastrointestinal tract, or umbilicus throughout the body. Laboratory data that supports this diagnosis consists of neutropenia, an increased number of bands,

and the presence of toxic changes such as basophilic cytoplasm and cytoplasmic vacuolization. In addition, hypoglycemia, azotemia, thrombocytopenia, or prolonged coagulation times may be seen.[3]

The diagnosis of neonatal sepsis can be difficult because there is no one single clinical presentation that is typical or one that consistently identifies a septic foal. Septicemia (systemic disease associated with the presence of bacteria in the bloodstream) and focal infection (i.e.,infection localized to lungs or joints) are two of the leading causes of morbidity and mortality in neonatal foals.

Septicemia can be diagnosed antemortem only by positive blood cultures which have an inherent 10 percent rate of false positives and as high as 25 percent false negatives.[4] Also, there is a delay of at least 24 hours and often up to 72 hours before culture and sensitivity results are confirmed. This holds up early specific therapy but necessitates vigorous initial therapy with an appropriate broad spectrum therapeutic antibiotic regime.

As stated previously, a definitive diagnosis of septicemia is made by bacterial culture of blood but also urine, cerebrospinal fluid, tracheal wash, and body cavity exudates.[5] These samples must be taken aseptically with best results achieved before antibiotic therapy started.

Although it is best to isolate and identify a specific causative agent and its' antibioticsensitivity pattern, this sometimes takes multiple samples and several days. Due to rapidity in which foal septicemias become fatal, antimicrobial therapy should not be delayed.

When treating the septic foal, antibiotic therapy should be instituted as early as possible. Before antibiotics can be started, intensive supportive care must begin to keep the foal alive. If the foal is hypothermic it should be put in a warm place with heating pads and blankets. Heat lamps can also be used. Care should be taken in the recumbent foal to prevent inhalation of debris (have the nose lower than the rest of the head) and also turn frequently to prevent hypostatic lung congestion, atelectasis, or development of pressure necrosis over bony prominences.[5]

The severity of illness will dictate the initial clinical approach to a critically ill septic foal. Intravenous fluid therapy should be instituted as early as possible. IV fluids should contain 5-10% dextrose in lactated ringers (sodium-containing replacement fluid). The volume of IV fluids will depend on the estimated deficit. The initial fluid flow rates can be as high as 500 to 1000 ml/hour in foals with severe hypotension.[2] In the weak or recumbent foal, fluid volume should be 80-120 ml/Kg/day (4 to 5 L in a 50 Kg foal) and given as continuous IV infusion.[5] The aim should be to replace 1/2 to 2/3 of the estimated deficit in the first 6 to 12 hours.[2] IV fluids should be warmed to normal body temperature.

Failure of a depressed foal to nurse can lead to severe hypoglycemia and decreased circulating fluid volumes. If a foal cannot nurse, a nasogastric tube can be placed and the foal fed mare's milk, goatmilk, or mare milk replacer at a rate of 8 to 10 mls/Kg of body weight every two to three hours (total of 4 to 5 L/day).[1] The nasogastric tube should not be left in for longer than 48 to 72 hours due to the potential development of pharyngitis, esophagitis, and gastrointestinal complications.[3]

Plasma therapy has also been used extensivly as preventative treatment of FPT and therapeutically to raise antibody levels in sick foals. Some indications for plasma therapy are failure of passive transfer, infectious conditions, high risk foals (prematurity, extremely stressed foals), and severe disseminated intravascular coaglopathy. It is difficult to know how much plasma is needed to raise the IgG level. In general, septic foals need 2-3 times the amount to raise the IgG by 200 mg/dl compared to a healthy foal being treated for FPT.[2] In a healthy foal, 1 liter/45Kg can be given IV over a 15 minute period. In sick foals, the rate should not exceed 2 liters/hour.[2]

Evaluation of acid-base status is important since hypoxemia can cause depression and lethargy as well as contributing to metabolic acidosis. A $pA0_2$ of less than 65 mmHg indicates hypoxemia and the need for oxygen therapy. The most common method of supplementing O_2 is via nasal insufflation.` Blood gases should be monitored closely (every 30-60 minutes), especially if the patient needs oxygen. The persistence of a base deficit > -8mEq/L even after volume replacement is grounds for bicarbonate administration.[2]
Calculation of bicarbonate required:

HCO3-(mEq)=BW(Kg) x 0.4(L/Kg) x base deficit(mEq/L)[2]

The rate of bicarbonate administration will vary with the degree of acidosis. Up to 1/2 of the calculated bicarbonate deficit can be given in the first hour. The rest of the bicarbonate is given over several hours while monitoring acid-base balance.[2]

Clinical laboratory values to assess response to therapy should be monitored closely. Adjustments in fluid volume, rate or composition should be made accordingly. The packed cell volume and total protein should be monitored twice daily. Increase in these are indications for more sodium containing fluids. The plasma electrolytes and bicarbonate should be monitored at least once daily. The body weight should be monitored daily. Foals on maintenance fluids receiving adequate nutritional support should have a small daily weight gain (1/2-2lbs). Significant weight loss indicates inadequate therapy with excessive fluid losses.[2] Urine output should be approximately at 3-4 L/day,[2] but not in excess of fluid intake in order to maintain normal glomerular filtration rate and renal function.

When deciding upon a certain antibiotic protocol it must be kept in mind that both gram-positive and gram-negative bacteria can cause septicemia in foals. As soon as sepsis is suspected, broad-spectrum antibiotics at correct doses should be started immediately. Bacteriocidal drugs are best because of the depressed condition of the immune system. When choosing a specific antibiotic for neonatal foal sepsis, several parameters must be kept in mind. Absorption of drugs given orally is erratic and may give unreliable tissue and plasma levels because of the continuous changes of the neonatal gastrointestinal tract during maturation.[5]

Compared with the adult horse who has 20-25% of total body weight in the extracellular fluid compartment (ECF), the foal has 40-50%. Since most antibiotics are in the ECF compartment, the volume of distribution would be greater and the concentration of plasma less in the foal than in an adult horse given the same dose/unit of body weight.[5] In addition, there may be less plasma protein binding of drugs in newborn foals, so a slightly higher dose of antibiotics may be warranted.[5]

One potential antibiotic therapy protocol is penicillin combined with an aminoglycosides. Aminoglycosides are eliminated by glomerular filtration and can accumulate in renal tissue and cause nephrotoxicity, especially if there is decreased renal perfusion.[7] The serum creatinine and urinalysis should be done periodically to check for granular casts and proteinuria (nephrotoxicity).

No matter what antibiotic protocol is chosen, antimicrobial therapy must be continued for at least one week after signs of recovery are apparent. Premature discontinuation may allow for development of foci of secondary infection or recurrence of septicemia.[5]

In foals up to 4 weeks of age localized and systemic sites of bacterial infection are very common. As stated previously, if the immunocompromised potentially septic foal is not treated immediately, the outcome is usually disastrous. The best treatment for neonatal sepsis is prevention. If this is not possible then early diagnosis and aggressive therapy is needed.

Several aspects of management can be carefully monitored and controlled in order to decrease the incidence and prevent neonatal sepsis. A good health care program for the brood mare and newborn foal is very important. A complete reproductive examshould be done and breeding history should be recorded. The nutrient requirements for the gestating mare should be monitored and she should also be immunized against equine herpesvirus I every 2-3 months during gestation and receive tetanus toxoid 30 days before parturition. A good deworming program should be adhered to along with pasture management. Poor management and dirty foaling areas predispose to foal sepsis.

Although all of these preventative measures are important, the most critical aspect is ensuring intake of adequate amounts of good quality colostrum. Even though the complete pathogenesis of neonatal foal septicemia is not completely understood, it is known that even under the best management practices sepsis does occur.

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